Amendments to the Specification:

Please replace paragraph [0002] with the following amended paragraph:

The present invention relates to endocrinology, physiology and pharmacology. The invention also relates to metabolic intervention with GLP-1 to therapeutically improve the function of ischemic and reperfused tissue. More particularly, it relates to methods and compositions for treating subjects suffering from intermittent claudication, typically associated with peripheral vascular disease (PVD).

Please insert before paragraph [0003] the following paragraphs:

Cellular damage to aerobic organ tissues is well recognized as a consequence of ischemia, whether endogenous as in the case of spontaneous coronary artery occlusion, or iatrogenic such as with open heart, coronary bypass surgery, or transplant procedures with the hear or other organs such as the lung, liver, kidney, pancreas and gastrointestinal tract. The degree and duration of the ischemia causing events are relevant to the amount of cell death and/or reversible cellular dysfunction. It is also known that much of the tissue damage in fact occurs upon reperfusion (i.e., resumption of blood flow) and re-oxygenation of the previously anoxic tissue.

As a side product of normal aerobic respiration, electrons are routinely lost from the mitochondrial electron transport chain. Such electrons can react with molecular oxygen to generate the reactive free radical superoxide which through other reaction steps in the presence of hydrogen peroxide and iron produces the extraordinarily reactive and toxic hydroxyl radical. Metabolically active aerobic tissues possess defense mechanisms dedicated to degrading toxic free radicals before these reactive oxygen species can interact with cellular organelles, enzymes, or DNA, the consequences of which could, without such protective mechanisms, be cell death. These defense mechanisms include the enzymes superoxide dismutase (SOD) which disproportionates superoxide, catalase which degrades hydrogen peroxide, and the peptide glutathione which is a non-specific free radical scavenger.

While not fully understood, it is believed that with ischemia of metabolic tissues and subsequent reperfusion, a complex group of events occurs. Initially during the ischemic period, intracellular anti-oxidant enzyme activity appears to decrease, including that of SOD, catalase, and glutathione. There is also an indication that the level of xanthine oxidase activity concomitantly increases in vascular endothelial tissue during the ischemic event. The combination of enhanced ability to produce oxygen free radicals (via enhanced xanthine oxidase activity) and reduced ability to scavenge the same oxygen radicals (via reduced SOD, catalase and glutathione activity) greatly sensitizes the ischemic cell to an oxidative burst, and hence damage, should these cells be subsequently reperfused with blood and therefore oxygen. This oxidative burst occurring within seconds to minutes of reperfusion could result in reversible and irreversible damage to endothelial cells and other cells constituting the ischemic-reperfused organ matrix.

Please insert before paragraph [0014] the following paragraph:

It, therefore, can be seen that there is a need for a safe effective composition having broad applicability to prevent or ameliorate the harmful effects of ischemia and reperfusion for tissues in general.

Please replace paragraph [0016] with the following paragraph:

The present invention relates to methods for treating or preventing intermittent claudication. In one embodiment, the method of this invention comprises administering to a subject a therapeutically effective amount of GLP-1 molecules. In another embodiment, the method comprises ameliorating, treating or preventing skeletal muscle injury caused by ischemia and/or reperfusion. In yet another embodiment, the method of the invention comprises promoting glucose transport into skeletal muscle. Individuals in need of treatment of ischemia and/or reperfusion are treated, preferably intravenously, with a composition which includes a compound which binds to a receptor for the glucagon-like peptide-1. Methods of the invention include a method for amelioration of skeletal muscle tissue injury caused by reperfusion of blood

flow following a period of ischemia, comprising administering to an individual an effective amount of a composition including a compound that binds to a receptor for glucagon-like peptide-1. Compositions of the invention may include a pharmaceutically acceptable carrier selected from the group consisting of saline, buffered saline, dextrose, water, glycerol, ethanol, lactose, phosphate, mannitol, arginine, treholose, and combinations thereof.

Please replace paragraph [0032] with the following paragraph:

As used herein, a "GLP-1 molecule" includes the following compounds. Mammalian GLP peptides and glucagon are encoded by the same gene. In the ileum, the precursor is processed into two major classes of GLP peptide hormones, namely GLP-1 and GLP-2. GLP-1(1-37) has the sequence: His-Asp-Glu-Phe-Glu-Arg-His-Ala-Glu-Gly-Thr-Phe-Thr-Ser-Asp-Val-Ser-Ser-Tyr-Leu-Glu-Gly-Gln-Ala-Ala-Lys-Glu-Phe-Ile-Ala-Trp-Leu-Val-Lys-Gly-Arg-Gly (SEQ ID NO:1). GLP-1(1-37) is amidated post-translationally to yield GLP-1(1-36)NH_2, which has the sequence: His-Asp-Glu-Phe-Glu-Arg-His-Ala-Glu-Gly-Thr-Phe-Thr-Ser-Asp-Val-Ser-Ser-T- yr-Leu-Glu-Gly-Gln-Ala-Ala-Lys-Glu-Phe-Ile-Ala-Trp-Leu-Val-Lys-Gly-Arg(NH_2) (SEQ ID NO: 2), or is enzymatically processed to yield GLP-1(7-37), which has the sequence: His-Ala-Glu-Gly-Thr-Phe-Thr-Ser-Asp-Val-Ser-Ser-Tyr-Leu-Glu-Gly-Gln-Ala-Ala-Lys-Glu-Phe-Ile-Ala-Trp-Leu-Val-Lys-Gly-Arg-Gly- (SEQ ID NO:3 [[NO 3]]). GLP-1(7-37) can also be amidated to yield GLP-1(7-36)amide, which has the sequence: His-Ala-Glu-Gly-Thr-Phe-Thr-Ser-Asp-Val-Ser-Ser-Tyr-Leu-Glu-Gly-Gln-Ala-Ala-Lys-Glu-Phe-Ile-Ala-Trp-Leu-Val-Lys-Gly-Arg(NH_2) (SEQ ID NO: 4). Likewise, GLP-1(1-36)amide can be processed to GLP-1(7-36)amide.